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L9	1410	L1 near8 activity	US-PGPUB; USPAT	OR	OFF	2007/04/04 15:33
L10	14374	(protease or peptidase or proteinase) near4 activity	US-PGPUB; USPAT	OR	OFF	2007/04/04 15:33
L11	47997	binding near4 activity	US-PGPUB; USPAT	OR	OFF	2007/04/04 15:33
L12	14554	(two hybrid)	US-PGPUB; USPAT	ADJ	OFF	2007/04/04 15:34
L13	14554	(two adj hybrid)	US-PGPUB; USPAT	ADJ	OFF	2007/04/04 15:34
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AN   2006:44325  CAPLUS
DN   145:139583
TI   Specific protein interaction of human Pag with Omi/HtrA2 and the
     activation of the protease activity of Omi/HtrA2
AU   Hong, Seung-Keun; Cha, Mee-Kyung; Kim, Il-Han
CS   Department of Biochemistry, Paichai University, Taejon, 302-735,
S. Korea
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SO Free Radical Biology & Medicine (2006), 40(2), 275-284
CODEN: FRBMEH; ISSN: 0891-5849
PB Elsevier
DT Journal
LA English
AB The human PAG gene product (hPag), one member of the TSA/AhpC family, is overexpressed by oxidative stress, which causes apoptosis. To investigate the apoptotic signal transduction mediated by hPag, hPag-binding protein was screened using the yeast two-hybrid system. Omi/HtrA2 was identified as the hPag-binding protein. Omi/HtrA2, a potent proapoptotic factor, is released from the mitochondria into the cytoplasm as the mature form showing serine protease activity during apoptosis in response to oxidative stress. We found that hPag was able to interact with the mature form of Omi/HtrA2, not with the precursor form of Omi/HtrA2. The binding of Omi/HtrA2 to hPag was shown to involve the PDZ-binding domain in Omi/HtrA2. Also, the carboxyl-terminal domain of hPag was shown to be critical for the protein interaction. Using the yeast two-hybrid system and in vitro binding assay, the reduced form of hPag was able to interact with Omi/HtrA2. Interestingly, the protease activity given by the mature form of Omi/HtrA2 was significantly activated by the binding to hPag. Taken together, these results suggest that the specific protein interaction may participate as a mol. switch in modulating cell death in response to oxidative stress.
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L8 ANSWER 2 OF 2 MEDLINE on STN DUPLICATE 1
AN 2004367968 MEDLINE
DN PubMed ID: 15101818
TI Binding of proteins to the PDZ domain regulates proteolytic activity of HtrA1 serine protease.
AU Murwantoko; Yano Masato; Ueta Yoshifumi; Murasaki Ai; Kanda Hidenobu; Oka Chio; Kawaichi Masashi
CS Division of Gene Function in Animals, Nara Institute of Science and Technology, 891605 Takayama, Ikoma, Nara 630-0101, Japan.

SO The Biochemical journal, (2004 Aug 1) Vol. 381, No. Pt 3, pp. 895-904.
Journal code: 2984726R. E-ISSN: 1470-8728.
CY England: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
LA English
FS Priority Journals
EM 200412
ED Entered STN: 25 Jul 2004
Last Updated on STN: 19 Dec 2004
Entered Medline: 10 Dec 2004
AB HtrA1, a member of the mammalian HtrA (high temperature requirement A)
serine protease family, has a highly conserved protease domain followed by a PDZ domain. Accumulating evidence has indicated that PDZ domains regulate protease activity of HtrA proteins. We searched for binding partners of the PDZ domain of mouse HtrA1 by yeast two-hybrid screening, and isolated proteins that were recognized by the HtrA1 PDZ domain through their C-terminal ends with a core consensus Phi-X-Phi-[V/L/F/A]-COOH sequence (where Phi is a hydrophobic/non-polar amino acid). C-propeptides of fibrillar collagens were most frequently isolated. Type III procollagen alpha1 C-propeptide, which was used as a model protein, was digested by HtrA1. HtrA1 cleavage of the collagen C-propeptide was enhanced by reductive denaturation of the C-propeptide and partly inhibited by removal of the C-terminal four amino acids from the C-propeptide, suggesting that the substrate recognition was facilitated by the binding of the free C-terminal ends of substrates to the PDZ domain of HtrA1. The synthetic oligopeptide (GM130Pep) that fitted the consensus recognition sequence bound to HtrA1 with a high affinity (K(d)=6.0 nM). GM130Pep stimulated HtrA1 protease activity 3- to 4-fold, but did not efficiently stimulate the activity of an HtrA1 mutant lacking the PDZ domain, supporting the notion that the PDZ domain enhances protease activity upon ligand binding . The peptide derived from Type III collagen alpha1 C-propeptide specifically stimulated protease activity of HtrA1, but did not stimulate nor significantly bind to HtrA2, suggesting that the collagen C-propeptide is a specific physiological regulator of HtrA1.

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